

LATENT HEART BLOCK

BY

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Received June 12, 1943

I have used the term "Latent Heart Block" to indicate a prolonged P-R interval without any manifest degree of block, such as dropped beats or 2 : 1 block.

This paper is a clinical study of a series of such cases, mainly from the point of view of the ætiology and significance of the condition. One might guess that latent heart block would be found in the same cases as, but at an earlier stage than, higher grades of heart block; and that a patient would progress from latent to dropped beats, next to 2 : 1, and finally to complete heart block. But such a regular sequence is not common.

Shortly, one can say that latent heart block is found in any type of heart disease at any age without much tendency to progress, and that there is a complete contrast with the well-recognized ætiology of complete heart block, which is atherosclerosis and high blood pressure in elderly patients. As might perhaps be expected, latent block is more often transient than is the case with complete block, but it is often persistent at a fairly steady level for long periods. It is also seen—sometimes to an unusual degree—in hearts that appear to be normal otherwise; here it may be an extreme instance of vagal activity (Fig. 1).

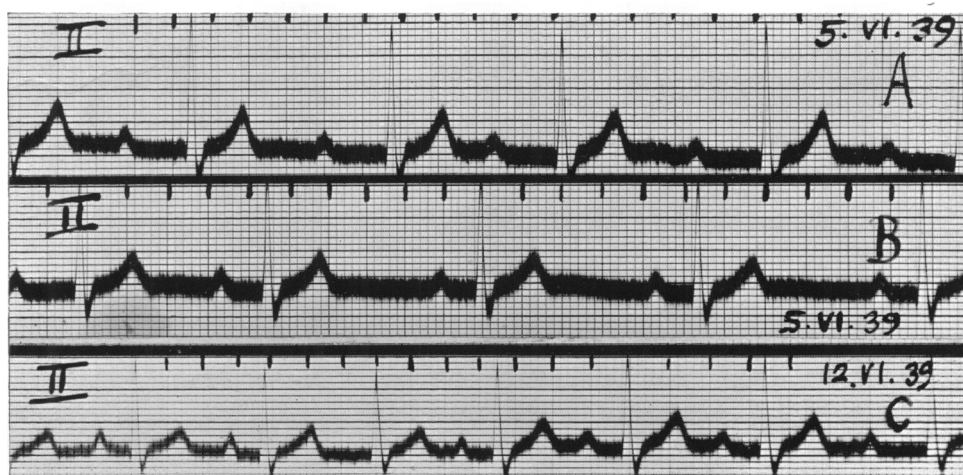


FIG. 1.—An unusual case of latent heart block, with longer and shorter P-R intervals without intermediate grades. From a young man without evidence of organic heart disease. Case 47.

(A) Longer P-R interval of 0.36 sec.

(B) Shorter P-R interval of 0.24 sec., except for the first response.

(C) Shorter P-R interval except for the last response and one instance of ventricular escape.

Of many plates available some showed a long or short P-R interval throughout all three leads, but most showed a change between the leads and some (mainly chosen for illustration) showed a change that was recorded while one lead was being taken.

In this and subsequent plates the time marker indicates fifths of a second, and generally these have been inked in at the top.

This lowest degree of heart block, when there is a prolonged conduction time without dropped beats or any higher degree of block, is, as a rule, only recognizable by graphic methods. Cowan and Ritchie (1922) said that it was not often observed and never caused any symptoms, although the patients in whom it occurred usually had obvious symptoms and signs of heart disease. Lewis (1920) mentioned two signs which might lead to its being suspected—a third sound in diastole owing to the auricular systole being separated from the ordinary ventricular sound, and in cases of mitral stenosis the murmur and thrill being isolated and concentrated at a special moment in diastole. These signs are not common, but the custom of taking more electrocardiograms has shown that the condition itself is common. It has therefore seemed worth investigating, and the term “latent” has been chosen as a convenient one for this variety of heart block. It is a true subdivision of heart block, although as a rule it is only made manifest by instrumental methods. The term incipient, which I suggested originally, seems less suitable because it might suggest that this early stage generally progresses sooner or later to a higher grade of heart block, and this is far from being true of most cases.

Any P-R interval of more than 0.20 sec. has been taken as prolonged, or more strictly one of 0.205 or above, as each has been recorded to the nearest hundredth of a second. Many cases with P-R intervals between 0.19 and 0.20 sec. have a conduction time that is prolonged and pathological for them, but unless cardiograms have been taken before there is no way of distinguishing the normal and abnormal at this level, and it is more practicable and more useful to accept the usual upper limit of 0.20 sec.

Chamberlain and Hay (1939) found the average P-R interval (0.16) 0.17–0.19 sec. in different age groups, but recorded a few normal cases where it was 0.22 sec. Hoskin and Jonescu (1940) in young women found most cases between 0.12–0.18 sec., but a very small proportion between 0.18–0.21 sec., the upper limit of normal originally given by Lewis and Gilder (1912).

The papers just quoted show that a few cases with a P-R of 0.21 or 0.22 sec. are normal, and the present investigation confirms their view; but the omission of all below 0.22 sec. would leave out so many pathological cases.

Paul White *et al.* (1941) have recently emphasized the differences often found in measuring the P-R interval in the different standard leads, that in lead II being generally the longest. This will be admitted by anyone measuring many cardiograms, and it would probably be best to work on a standard taken from lead III, as they suggest. But these cases were seen before realizing the extent of this difference or reading his paper, and generally where there were differences an average figure had been taken. In any case, it would have little effect on the very considerable prolongation found in most of these patients, as only 4 per cent of their series showed a variation up to 0.02 sec.

The present series of cases was obtained by looking through 10,000 consecutive electrocardiograms taken at Guy's Hospital in the twelve years 1927–38 (172 cases) and adding all others (61 cases) I had remembered to index from those seen at the Heart Hospital and in private: the latter would fail to include many with only a slight increase in the conduction time, but few of much interest where it was longer; the former would provide an unselected consecutive series, as when there seemed any doubt the P-R interval was measured.

Ten cases with congenital complete heart block have been omitted as they form a special type that has been reported and discussed elsewhere (Campbell and Suzman, 1934a).

This means that about 2 per cent of all patients sent to a cardiac department show latent heart block and rather more than 1 per cent show some clinical degree of heart block.

The 141 cases with latent heart block only have been divided into three grades; (IV A) 31 cases with a P-R interval of 0.26 sec. and above, (IV B) 45 cases with a P-R interval of

from 0.23 to 0.25 sec., and (IV C) 65 cases with a P-R interval above 0.20 but not more than 0.22 sec.

TABLE I

NUMBER OF PATIENTS WITH LATENT AND HIGHER GRADES OF HEART BLOCK

	Guy's Hospital	Heart hospital and private	Total
Number of electrocardiograms	10,000	—	—
Total number of patients	5,320	—	—
Number with some degree of heart block	172	61	233
I. Complete and/or 2 : 1 block without latent	25	11	36
II. Latent and complete or 2 : 1 heart block	15	12	27
III. Latent heart block and dropped beats	17	12	29
IV. Latent heart block only	115	26*	141
A. P-R, 0.26 sec. and above	23	8	31
B. P-R, 0.23 to 0.25 sec.	34	11	45
C. P-R, 0.20 to 0.22 sec.	58	7	65

* This figure is relatively low because they were not searched for systematically, as were the Guy's cases.

ÆTIOLOGY OF CASES WITH HEART BLOCK

The ætiology of cases with latent heart block is summarized in Table II, and for comparison a series of cases, (I) with complete and/or 2 : 1 heart block *without* latent block, (II) with complete and/or 2 : 1 heart block *and* latent block, and (III) with dropped beats but no higher degree of block, have been analysed and tabulated in the same way. It was found that these three grades and the three grades of latent heart block (IV A, IV B, and IV C) could, as far as ætiology is concerned, be combined into three larger groups.

Those with a P-R interval from 0.20 to 0.22 sec. and those with a P-R from 0.23 to 0.25 sec. were combined because they showed no apparent difference, except that (as might be expected) there were among the former more cases with normal hearts and rather more with thyrotoxic hearts. On the other hand, those with a P-R interval from 0.26 sec. upwards showed a much higher proportion of cases with rheumatic fever and acute infections and a much smaller proportion with thyrotoxic or normal hearts. This was also the case with the patients who showed dropped beats, so these were combined into a second group.

Finally there was little ætiological difference between those with 2 : 1 and/or complete heart block, whether they had or had not also latent block, so these were also combined into a third group.

The figures for these three larger groups are expressed as percentages in Table II, after combining some of the smaller ætiological subdivisions to make comparison as easy as possible. The differences are most striking. In the first group with the least prolonged P-R intervals, up to 0.25 sec., 24 per cent had thyrotoxicosis or no apparent heart disease, both these factors becoming much less significant in the second group and absent in the third group with 2 : 1 or complete heart block. Chronic rheumatic heart disease was also of importance in the first group, being the ætiological factor in 22 per cent; this figure falling to 12 per cent in the second, and to 3 per cent in the third group.

In the second group, with the longer P-R intervals of 0.26 and above or with dropped beats, 41 per cent had active rheumatic fever or other acute infections, while this factor was responsible for only 11 per cent of the first group and for only 2 per cent of the third. No other ætiological factor showed a significantly larger figure in this group than in the first group with a shorter P-R interval.

In the third group with 2 : 1 or complete heart block, there was an even more striking contrast: 69 per cent of the cases, against 29 and 23 per cent in the first and second groups, had myocardial disease as the ætiological factor. Syphilitic heart disease was also much more

prominent in this group, being found in 9 per cent against the insignificant figure of 2 per cent in the two former groups. Primary myocardial disease with syphilitic and hyperpietic heart disease were together responsible for 95 per cent of the cases with 2 : 1 or complete heart block, while in the two former groups these three factors combined were not responsible for more than 43 and 40 per cent. Hyperpietic heart disease alone had a much more regular incidence in the three groups, varying only from 12 to 17 per cent. Further subdivision of the myocardial cases did not reveal any striking differences.

TABLE II
ÆTIOLOGY OF DIFFERENT GRADES OF HEART BLOCK (PERCENTAGES)

	IV B and IV C P-R, 0.20 to 0.25 sec.	IVA and III P-R, above 0.26 or with dropped beats	II and I Complete and 2 : 1 heart block with or without latent block
Tonsillitis and acute rheumatism	11	41	2
Chronic rheumatism	22	12	3
Syphilitic	2	2	9
Hyperpietic	12	15	17
Myocardial	29	23	69
Thyrotoxic	13	2	0
No apparent disease	11	5	0
Total numbers	110	60	63

ÆTIOLOGY OF CASES WITH LATENT HEART BLOCK

There were 110 cases with a P-R interval from 0.20 to 0.25 sec.; few of these call for individual comment except that the presumed normals will be enumerated. Otherwise, the diseases from which they suffered seemed to be much the same as might be expected in any other collection of hospital cases.

Of the 110, 48 belonged to the myocardial group (about equal numbers having raised blood pressure or disease of the coronary arteries or heart failure or a large heart without any of these causes being obvious), 36 to the rheumatic group, 14 to the thyrotoxic group, and 12 to the normal group.

There were 31 cases, where the P-R interval was 0.26 sec. or more. Acute rheumatism or tonsillitis or treatment with digitalis were more often responsible for the longer intervals, and in some cases these were the only cause of the P-R interval being prolonged, but in others there was already some degree of prolongation that was increased by a transient infection or by treatment with digitalis. These greatly prolonged P-R intervals will be considered in more detail later (see p. 175). First, however, certain general points about the ætiological groups will be discussed.

Acute rheumatism. Little need be said about this as the prolonged conduction time of acute rheumatism is well known. Parkinson, Gunson, and Gosse (1920) found some degree of block in 30 per cent of their cases during the acute stage. Cohen and Swift (1924) found it over 0.21 sec. in 22 per cent of their cases and somewhat prolonged in 84 per cent, if any increase of more than 0.02 sec. above the normal for that patient was counted as pathological. In a recent study, Keith (1938) found this small increase in 80 per cent of his cases with clinical evidence of rheumatic carditis; he did not say how many were over 0.20 sec., but as the average figure was up to 0.18, a good many must have been so. He brings forward good evidence that the increase is due to vagal activity and, though his title of "over-stimulation of the vagus nerve" might suggest some central factor which would hardly fit in with the general clinical picture, he suggests that the part involved is the vagal nerve terminations in the heart.

All are agreed about the relative rarity of manifest block in these rheumatic cases: Carey Coombs (1924) had only come across six cases where dropped beats were produced by acute rheumatism.

Examples have already been described (Campbell, 1943) and are pertinent in this connection also, as soon after the dropped beats they had latent block alone. In fact, my own records show few instances of classical rheumatic fever as a cause of latent block only and it was more often recorded after a minor relapse, but this is because cardiograms of patients with rheumatic fever were not taken as a routine at Guy's Hospital, but only when there was some special reason.

The return of the P-R interval to normal may be very quick and complete, even after a severe attack. A boy, aged 18, was admitted with a classical attack and had an irregular heart with dropped beats, generally after P-R intervals of 0.25 and 0.37 sec.; after a week it had fallen to 0.18 sec. (Case 13). Here there was little correspondence between the sedimentation rate and the increase of conduction time, for after six weeks, when the sedimentation rate had only dropped from 98 to 47, the P-R interval was down to 0.16 sec. In a man with a very severe attack, the P-R interval was increased from 0.16 to 0.34 sec. (with some dropped beats that were not graphically recorded); three days later it was 0.25 and a week after this 0.19, and after another week 0.17 sec. (Case 22). In a younger man with an attack of average severity it fell from 0.32 to 0.19 sec. in 18 days (Case 54). These three patients all made good recoveries as regards their heart, and a quick recovery in the conduction time is certainly one favourable sign for a good outlook as regards the heart.

Sometimes the P-R interval is increased considerably from what appear to be very minor relapses. In a young man with mitral and aortic disease, it rose during convalescence from 0.20 to 0.26 sec. with a very minor return of pain and was still 0.24 a month later (Case 31). A girl was convalescent after an attack of chorea and slight carditis, and her sedimentation rate had fallen to 6: her P-R interval increased from 0.21 to 0.28 sec. with no other evidence of a relapse except a rise of heart rate from 80 to 90 (Case 52).

In two severe cases the increase of conduction time was no more than in these minor ones. In a boy of 17 with mitral and aortic disease, where it had been about 0.21 for many years, it rose to 0.28 and, finally, to 0.31 sec. (with associated S-T inversion in leads II and III) in his terminal attack of rheumatic fever (Case 36). In a girl with rheumatic aortic regurgitation, where the P-R interval was generally about 0.21, it did not rise above 0.25 sec. in a recurrent attack with pericarditis that was severe enough to produce fairly deep S-T inversion (Case 85).

It is obvious even from this small number of cases that the increased conduction time is not an accurate measure of the severity of the attack or even of the cardiac involvement. Nevertheless, the quick fall towards or to normal is a favourable sign, and often the changes in heart rate, the sedimentation rate, and the conduction time do seem roughly parallel, so that any one is in that case a good measure of the severity of the attack.

Other acute infections are dealt with later.

Chronic rheumatic heart disease. Quite apart from the temporary effect of acute carditis there may be permanent lengthening of the P-R interval as a result of old rheumatic infection.

Typical examples, all of whom had mitral stenosis, were a woman with a P-R interval between 0.23 and 0.26 sec. during four years; a girl of 8 with a P-R interval of 0.23 sec. for three years; a woman of 55 with a P-R interval of 0.23 sec., who was known to have had this for eight years; a woman of 33 with a P-R interval of 0.23 sec., a large P II, and cyanosis of unusual degree (like Case 45), who died a few months later; and a man of 29 with a P-R interval of 0.24 sec., who had paroxysmal auricular tachycardia at a rate of 146 without any change in the P-R interval.

As a rule the lengthening was not extreme, but there was one striking exception—a girl with mitral stenosis who was under regular observation for eight years with a P-R interval that increased gradually from 0.26 to 0.36 sec.; apart from tachycardia there was no evidence of any return of active carditis, unless a silent carditis precipitated failure in the last two years of her life (Case 45; see Appendix).

In many of the cases with a long P-R interval due to active rheumatism some slight prolongation had been noticed even before the infection.

There were six patients, who had latent heart block when first seen and later developed auricular fibrillation. One girl with rheumatic aortic incompetence, whose P-R interval had been raised from its normal 0.21 to 0.31 sec. by unwise digitalization during recurrent subacute rheumatism, developed fibrillation four years later. At least three of the others had mitral stenosis; a girl, who had rheumatic fever when 4, had a P-R interval of 0.21 when 10, and fibrillation when 15; a young woman who had a P-R interval of 0.22 sec. during the fifth month of her pregnancy, developed fibrillation after her confinement; and a woman of 37 who had gradually been forced to restrict her very active life, was seen with bronchitis, a P-R interval of 0.24 sec., and a very large P II, and five months later developed fibrillation. No recurrence of active rheumatism was suspected in these last two, but it is, of course, one exciting cause of the onset of fibrillation in some cases.

In addition, two cases first seen with fibrillation had long P-R intervals when normal rhythm was restored with quinidine; in one of these, normal rhythm continued for some years and the P-R

interval was 0.24 sec., so no question of active infection arose. In one man with flutter a long P-R had been enough to produce a heart rate of 72 owing to 4 : 1 block (Case 41, see Appendix), but another in whom the P-R interval was 0.24 sec. had had fairly rapid flutter simulating fibrillation before it was stopped.

In such a mixed group of patients, it is not easy to know the prognosis, and at the time I had not regarded latent heart block as a specially unfavourable sign, perhaps because in those who had done badly there were often other unfavourable signs. Looking through the cases as a whole, it seemed to me they had done less well than might have been expected. The finding of a long P-R interval of 0.24 or above should, therefore, lead to a very careful review of the patient's life and activity and these should be limited until further observation allows gradual progress. No doubt it is partly because latent heart block and large P waves are often found in hearts that are badly damaged in other ways, but it may be that sometimes latent block is a sign of a rheumatic infection that is otherwise silent, and this possibility should be carefully considered.

Myocardial and hyperpietic disease. There seems little special to say about these cases. It was not regarded as a finding of special interest or significance, so no great effort was made to follow them up, but it was often noted that a P-R interval of 0.21–0.24 sec. remained stationary for one or two years. Coronary atheroma (with coronary thrombosis or simple angina), hyperpiesis, congestive heart failure—alone or in combination—or an enlarged heart without evidence of these other factors were all common.

Rarely did any patient, found by chance to have latent heart block, develop complete block. But there were several instances where a patient, sent up because Stokes-Adams attacks or heart block were already suspected had, when examined, latent block only, and later was seen again when he had a higher degree of block. Such cases with 2 : 1 or complete block will be dealt with in a subsequent paper and the relationship of these to latent block will then be discussed more fully. One old man included in this series was sent up with a history of Stokes-Adams attacks and was found to have latent block only (Case 58; see Appendix), but probably if he had been seen more often, complete heart block would have been recorded as he died two years later in another Stokes-Adams attack. In all other instances (unless Case 59 is another exception) such patients were seen at other times with higher grades of block and will therefore be dealt with in the later paper.

Case 55 (Appendix) was of interest, both because of his P-R of 0.35 sec. and because he had angular pain in a phantom arm.

Case 44 (Appendix) would have been included in the group with no heart disease and the change attributed to his obesity, if he had not been followed up; ten years later the blood pressure was 180/120, but it is possible that this was a coincidence as the P-R interval, though still long, was shorter (Fig. 2).

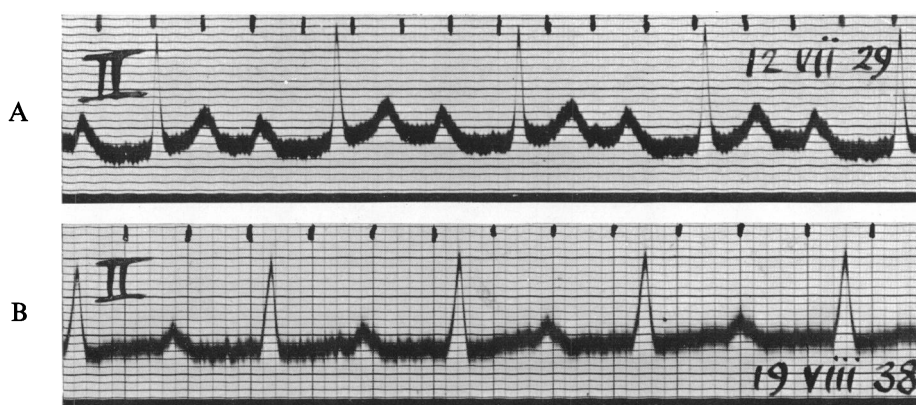


FIG. 2.—Latent heart block with obesity from a man, aged 40, in 1929. Case 44.

(A) 1929, P-R of 0.36 sec. with blood pressure 130/80.

(B) 1938, P-R of 0.31 sec. with blood pressure 180/120.

Bundle branch block. The association of this with latent block was much more common than would be expected if it were a chance coincidence. Apart from the patients with higher grades of block and B.B.BI., there were 5 of the 141 with latent block who had B.B.BI. also (2 of the 31 with a P-R interval of 0.26 sec. or above).

This led to my looking through 25 consecutive cases of bundle branch block, excluding any that were already in this series. Sometimes it was not easy to measure P-R, because it was difficult to know where QRS began, and this led to even greater differences than usual between the measurements in the different leads. Using lead II, where it often seemed the longest, the average P-R interval was 0.17 sec.: in 7 it was from 0.12 to 0.15 sec.; in 11 it was from 0.16 to 0.19; in 6 it was 0.20 sec. or a little more; and in 1 only was it as long as 0.24 sec.

Thyrotoxicosis. Nearly one tenth of all the cases with latent block had thyrotoxicosis, but a very large number of thyrotoxic patients were sent for cardiograms. In my own cases where cardiograms had been taken from time to time over a longer period, the P-R interval was not often lengthened without an added infection, so the high proportion may have been due to the large amounts of Lugol's iodine, which many of these patients were taking before operation. This was not the whole explanation, for sometimes it was seen when the patient was not having iodine, and in one case the P-R interval was 0.22 sec. when she was admitted for her first operation and 0.14 sec. six months later, when she was improved but not yet well and was having a second operation: on both occasions she was having Lugol's iodine.

Myxædema. There were also three cases with latent heart block and myxædema. In one the P-R interval was 0.23 sec. when there were low-voltage or flat T waves and only 0.20 when the T waves were upright after thyroid treatment. In another the P-R interval was 0.23 without thyroid, and 0.21 six years later when she had again been without thyroid for several weeks. I was surprised at these findings as some years ago in reporting the cardiographic changes in eight cases of myxædema (Campbell and Suzman, 1934b) comment had been made on the low voltage P that increased with thyroid treatment, but not on the P-R interval. In five of the eight cases there was no significant change. However, three of the eight showed a P-R becoming shorter by about 0.03 sec. after thyroid treatment—0.24 to 0.20 sec., 0.21 to 0.18 sec., and 0.19 to 0.16 sec.—so that this should be added as one of the fairly common features of the myxædema heart.

Congenital heart disease. Among the cases with latent block there were three examples of this. A girl, aged 13, with coarctation of the aorta and a patent ductus arteriosus, had a P-R interval of 0.26 sec.; she died a year later. A man, aged 28, with bicuspid aortic valves, great enlargement of the heart, and congestive failure had a P-R interval of 0.24 sec.; he died six months later. A woman, aged 35, with moderate enlargement of the heart and an auricular septal defect had a P-R interval of 0.23 sec.; she was alive three years later but was troubled with hæmoptysis.

It seems likely that in all these the latent block was a subsequent development and not part of the original congenital condition, for they were all sick people.

Trauma. There was no cases in this series where latent block seemed to be due to trauma, but such have been reported. One has been added recently by Barber (1942) where a P-R interval of 0.35 sec. persisted for at least five months. Dr. J. R. B. Hern has shown me an example of long P-R, 0.24 sec., following blast from a bomb: though she had orthopnoea and œdema of the legs, the main damage was at first thought to be pulmonary and this was supported by the X-ray findings and the continued cough; however, a year later the P-R interval was still 0.24 sec.

Diphtheria. No cases have been included as diphtheritic. One woman, aged 42, may be mentioned as a possible instance; seen because of extrasystoles, she had a P-R interval of 0.26 sec. and some cardiac enlargement, and a history of diphtheria as a child (*Case 34*). One girl with a severe relapse of thyrotoxicosis had a P-R interval of 0.22 sec. from the third to the eighth month after diphtheria, but its duration before her attack was not known. A past history of diphtheria has been mentioned in some others (*Cases 47, 49, and 55*), but in none was there evidence that the prolongation dated from that attack, and it may have represented no more than the expected incidence of past diphtheria in any community.

Bruce Perry (1939) has mentioned two cases of complete heart block persisting for several years after diphtheria, but this is certainly rare. Jones and White (1928) investigated 100 patients who had suffered from diphtheria at least five years before; in 70 the attacks had been severe. In no case could they find any evidence that any chronic heart disease had been produced, and on electrocardiographic examination there was nothing more than one example of nodal premature beats with ventricular escape.

Other acute infections. Tonsillitis seemed the most important infection after acute rheumatism. Sometimes it was difficult to be sure that an apparent attack of tonsillitis was not really rheumatic, as illustrated by the following example.

Case 43. A boy of 15, with a doubtful history of growing pains when he was 4, was seen a few days after a sore throat that had followed a 100-mile cycle ride. He was pale and thin, with red tonsils, but no signs in his heart except a P-R interval of 0.26 sec. After his admission to hospital, the sore throat recurred with very little rise of temperature, but he was found to have several rheumatic nodules, and later had one attack of paroxysmal tachycardia. During the month in hospital the P-R interval fell to 0.21 sec.

In spite of this difficulty I feel satisfied that in many cases there were no grounds for suspecting a rheumatic infection. Two have already been quoted where dropped beats and afterwards latent block followed tonsillitis that did not appear to be rheumatic (Cases 5 and 17). The latter has been under observation for 10 years without any evidence to suggest rheumatism, although her P-R interval rose to 0.32 sec. after a transient sore throat, quickly returning to 0.16, at which level it has remained for 10 years. The two patients that follow are further examples without any evidence of rheumatism.

Case 56. A healthy-looking boy, aged 14, came to hospital ten days after an attack of tonsillitis that had lasted three days. He had no history of rheumatism and no symptoms except occasional faintness during the past two years. Nothing was found on examination except a P-R interval of 0.27 sec. and two weeks later this had fallen to 0.21 sec.

Case 59. A boy, aged 18, who had a Stokes-Adams attack three weeks after tonsillitis and came to hospital four days later with a P-R interval of 0.28 (Fig. 3), that quickly fell to 0.16, is reported in the appendix. There was nothing to suggest that the illness was really diphtheria.

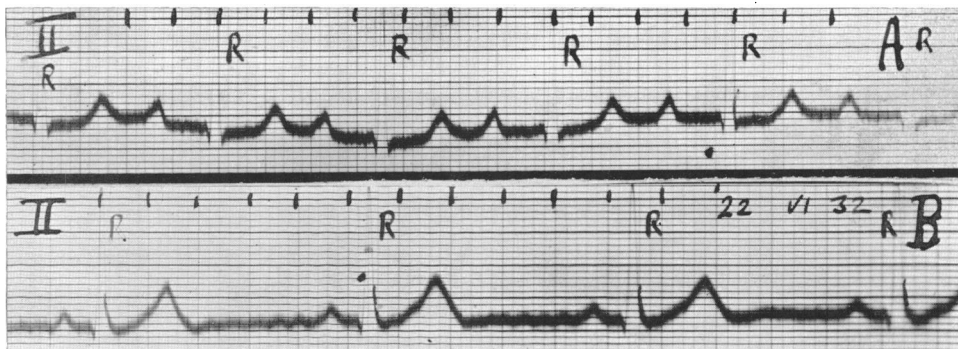


FIG. 3.—Temporary latent heart block after tonsillitis from a young man whose heart was otherwise normal. Case 59.

- (A) Lengthened P-R interval, 0.28 sec., three weeks after an attack of tonsillitis and four days after a Stokes-Adams attack.
 (B) Rapid return of the P-R interval to normal, 0.16 to 0.17 sec. This curve was taken five weeks after (A), but the P-R interval was 0.18 sec. a week after, and 0.16 to 0.17 two weeks after (A).

Two other patients were included in this group as one was in hospital with a sore throat and the other came up soon after one (Cases 42 and 47; see Appendix). But there was not the same speedy fall of the P-R interval, and follow-up study after 1 and 5 years respectively showed that they still had equally long P-R intervals. This and some other unusual features suggest that the latent heart block may really have preceded their tonsillitis and that they ought to be included as cases without any evidence of heart disease. If so, the range of possible variation in the P-R interval under vagal influence must be considerably widened.

CASES WITH NO APPARENT HEART DISEASE

9 of the 65 cases with a P-R interval between 0.20 and 0.22 sec. were regarded as having no heart disease, though in each case some question had been raised that led to their examination. They were as follows: a girl of 17 with habit spasm and a girl of 19 with obesity; the remainder were men, three being under 40 (31, 28, 27) with pleurisy, an anxiety state, and sinus bradycardia with sinus arrhythmia; three being between 40 and 50 (42, 48, 49) with extrasystoles, vaso-vagal attacks, and a duodenal ulcer; and the ninth, a man of 67 with gallstones. It is possible, of course, that some minor degree of heart disease escaped detection in these cases, but it seems more likely that a P-R interval of 0.21 or even 0.22 sec., though unusual, is not outside the limits of normal.

Case 94. A man, aged 27, who was only seen because of the slow and irregular heart found when he was up for life insurance, was of interest because he was probably an instance of the effect of vagal activity. His heart rate varied between 35 and 48 and his P-R interval was 0.22 sec.

There were 3 of the 45 patients with a P-R interval of from 0.23 to 0.25 sec., who were thought to have normal hearts, and these must be considered in a little more detail.

Case 84. A girl of 21, with a diagnosis of hysterical fits, was sent for examination of her heart because she had twice had chorea, the last time four years before. The P-R interval of 0.24 sec. may have been the result of her attacks of chorea, but nothing abnormal was found about her heart.

Case 72. A man, aged 39, had been under observation for some years with duodenal ulcer and had done well. On one occasion at out-patients, he complained of dyspnoea, and though nothing else was found, his P-R interval was 0.25 sec. On later occasions it had reverted to 0.20 sec., so probably this was due to some transient infection in a heart that was otherwise normal. If so, he should of course be placed in the group with acute infections, but in the absence of more definite evidence it seemed best to discuss him here.

Case 77. A man, aged 50, had fairly severe bronchial asthma of long standing. His heart was thought to be normal but the P-R interval varied between 0.23 and 0.25 sec. It is possible that this may have been the one early sign of heart disease that was not detected otherwise.

In none of these three cases was there any other sign of heart disease.

The main difference between these two groups is that in those with a P-R interval of from 0.20 to 0.22 sec. there was little or no reason for suspecting the heart, while in the group of possible normals with a P-R interval of between 0.23 and 0.25 sec. there was, in every case, some suspicion of early heart disease, though the latent block was the only certain evidence.

One would not expect to find any patients with normal hearts with a P-R interval of 0.26 sec. or more, but three must be mentioned as other evidence of heart disease was trivial or absent.

Case 51. A man, aged 41, was thought to have a normal heart, though at first there was some doubt if the dyspnoea of which he complained might possibly be post-influenzal. In 1931 his P-R interval was 0.24 sec. In 1934 I got him to resume work, and in 1939 his symptoms were much the same and certainly no worse; the P-R interval was 0.27 sec. and records taken most years suggested that this really had been a gradual but irregular increase. His heart was not enlarged and there was no other evidence of disease.

It is of interest because it is possible that it represents a slight progressive change that in the course of several more years will result in a higher grade of heart block recognizable clinically. But this course is unlikely for many of these cases have been followed for years, and such changes have been looked for and yet we have no other similar example.

Case 40. Here, the interpretation is difficult. In 1927, when 43, he was thought to have a P-R interval of 0.35 sec. (Fig. 4). He complained of extrasystoles, paroxysmal tachycardia, and symptoms that were due to anxiety. He was seen at intervals during the next ten years, and never had any symptoms suggesting heart disease, nor a P-R interval longer than 0.16 sec. He was not taking any drug known to affect the heart at the time the long interval was observed.

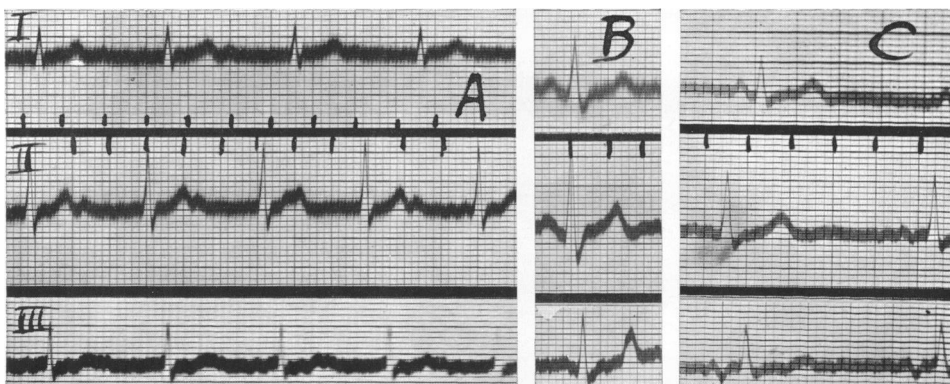


FIG. 4.—Temporary latent heart block of high grade. Case 40.

- (A) Cardiogram of 1927, with a P-R interval of 0.35 sec. No other explanation seems possible, especially in view of the fourth response in lead II being premature, but still with the same P-R interval.
 (B) A week later, and (C) ten years later, both these showing a normal P-R intervals of 0.16 sec.

It is possible that this was an unusual result of a minor transient infection; although an extreme example it would not be much more extreme than some others.

Case 49. A man, aged 28, was sent for a cardiogram because of extrasystoles. The diagnosis of tuberculosis of the lungs was considered but not proved, for though he complained of cough and loss of weight, the X-ray was inconclusive. His heart was thought to be normal except for the extrasystoles, which sometimes after exercise were so frequent as to simulate paroxysmal auricular fibrillation, and a variable P-R interval which changed from 0.20 to as long as 0.36 sec. There was no gradual increase, but on two occasions where records were obtained (and as far as could be judged on several other occasions) there was suddenly a very long P-R interval up to 0.36 sec. with the P-P interval remaining constant, and the R-R interval lengthening appreciably (Fig. 5). Such a

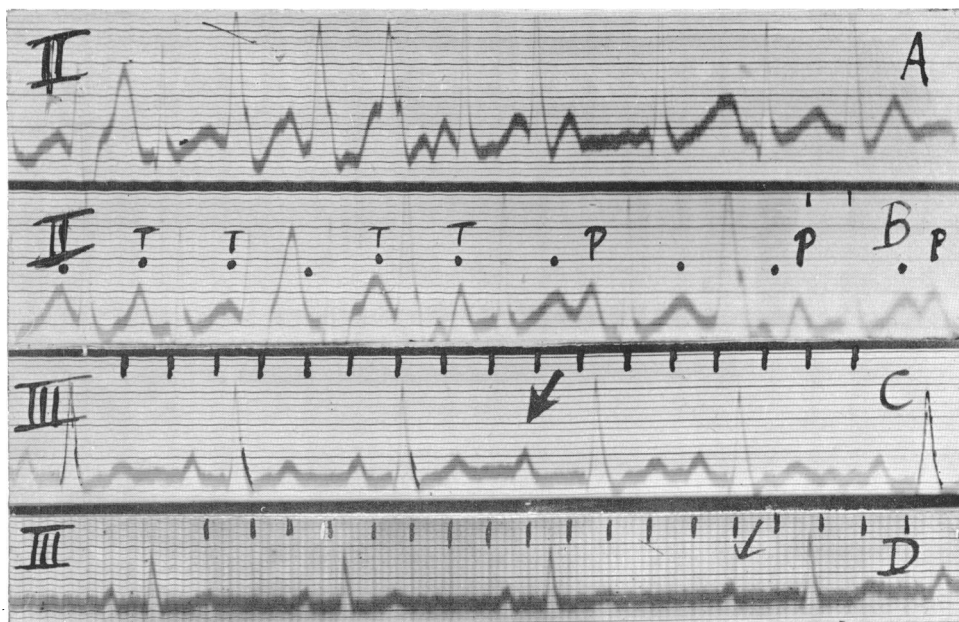


FIG. 5.—Irregular latent heart block of high grade. Case 49.

(A) and (B) Irregular rhythm after exercise.

(C) April 1935 and (D) June 1935, showing single P-R intervals of 0.36 sec., interrupting P-R intervals of 0.20 sec. These are probably vagal effects in a normal heart.

sudden change must almost certainly be a vagal effect and related to sinus arrhythmia, but it is curious that even so it should not occur more gradually. Nor was there any sinus bradycardia such as has been described in Case 94 with much less lengthening of the P-R interval. After eight years he was written for and seen again. He had been getting on well and had been working regularly, with no symptoms. His heart rate was 80 with much irregularity after exercise; this seemed to be (a) an extreme sinus arrhythmia, the rate doubling, but not suddenly as with S-A block, and at times; (b) a gross irregularity that was not influenced by respiration and lasted for some beats only. X-ray confirmed that there was no enlargement of the heart and showed some old calcified areas below the right clavicle.

Cases 42 and 47 in the appendix should be noted in this connection; at the time the latent block was thought to be the result of attacks of tonsillitis, but its persistence and some other features suggest that in these two as well as in *Case 49* it may really have been an unusual vagal effect.

THE LENGTH OF THE P-R INTERVAL

As would be expected, P-R intervals a little above the normal are very much more common than those greatly above it. There were a large but diminishing number at each hundredth of a second from 0.21 to 0.24, a moderate but more slowly diminishing number at from 0.25 to 0.28, and a smaller and fairly steady number (at each hundredth of a second) from 0.29 to 0.37 sec. Those with P-R intervals of 0.21 and 0.22 and those with P-R intervals of 0.23 and 0.24 have been combined, and so on, partly because the numbers were small and partly because there was a little tendency to record the measurements by even numbers. The full figures are shown in the second column of Table III.

TABLE III

LENGTH OF THE P-R INTERVAL

Length of P-R interval in seconds	Number of cases			Total
	(IV) Latent heart block only	(III) Dropped beats and, at other times, latent block	(II) Complete or 2 : 1 block and, at other times, latent block	
Less than 0.20 sec.	—	2	5	7
0.21 0.22	65	4	5	74
0.23 0.24	39	1	6	46
0.25 0.26	13	3	3	19
0.27 0.28	8	6	1	15
0.29 0.30	2	3	2	7
0.31 0.32	4	4	1	9
0.33 0.34	3	2	2	7
0.35 0.36	4	1	0	5
0.37 0.38	2	0	0	2
0.39 0.40	1	1	0	2
0.44	—	0	1	1
0.47	—	1	—	1
0.56	—	1	—	1
Total number	141	29	26	196
Average P-R interval ..	0.24 sec.	0.28 sec.	0.25 sec.	

A smoothed curve has been drawn in Fig. 6. It falls very sharply at first between 0.24 and 0.25, but then levels out and shows little further tendency to fall after 0.29 until it practically comes to an end at 0.40 sec., above which there were only single cases.

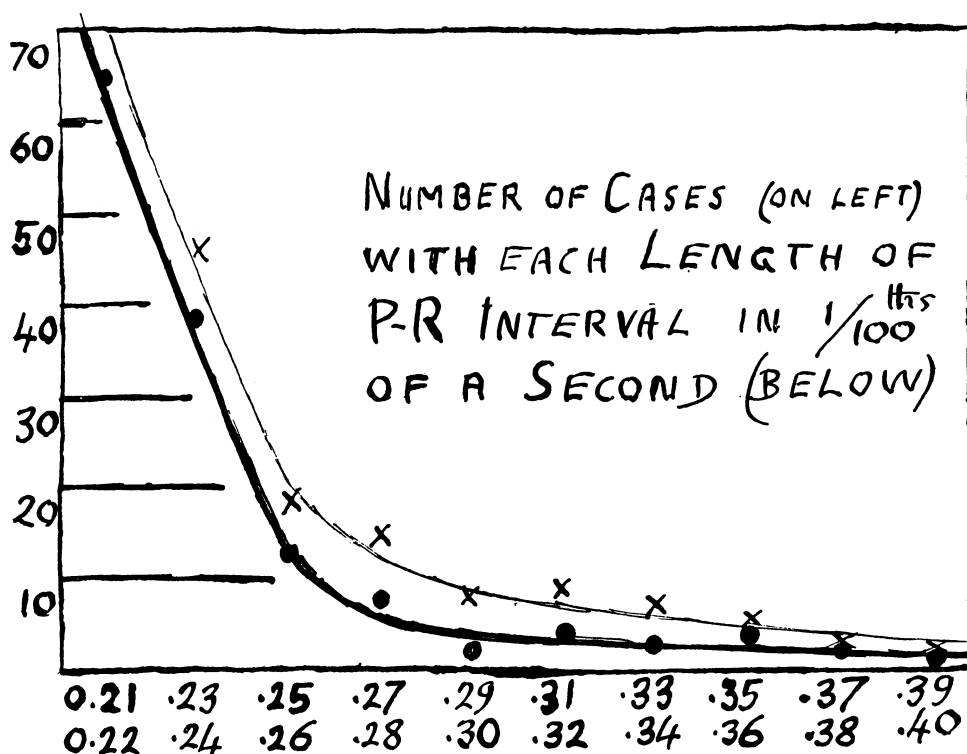


FIG. 6.—Number of cases with latent heart block.

Abscissæ, length of the P-R interval in hundredths of a second; ordinates, number of cases.

The thick line below includes the 141 cases with latent heart block only.

The lighter line above includes the additional 53 cases that had dropped beats or higher grades of heart block at one time and latent block only.

There were 141 cases with heart block that was latent only; 29 others had sometimes dropped beats and sometimes latent block; and another 26 had sometimes 2 : 1 and/or complete heart block and sometimes latent block. One might expect in each of these groups also, either that the frequency distribution would be similar or possibly that the length of the P-R interval would vary round a much higher average figure.

This was not so; in those with latent heart block only, 74 per cent of the cases had a P-R interval of 0.24 sec. or less (46 per cent, 0.21 or 0.22); in those with dropped beats, only 24 per cent had a P-R interval of 0.24 or less, and 65 per cent had figures of 0.27 sec. or above; those with 2 : 1 and/or complete heart block also, was more like the former than the latter, 61 per cent having a P-R interval of 0.24 or less, but being more equally distributed through this range.

In those with latent block, the average P-R interval was 0.24 sec. and most of the cases were between 0.21 and 0.24 sec. In those with dropped beats, the average was much higher, 0.28, and most of the cases (55 per cent) were between 0.27 and 0.34 sec. In those who had at times higher grades of heart block the average was intermediate and nearer to that of latent block, viz. 0.25, with a range more equally distributed between 0.19 and 0.26 sec.

The full figures have been given in Table III. Table IV shows certain statistical data which may make the results clearer to some. The median is the point above and below which there are equal numbers of cases; the mode is the point at which the largest number of cases are grouped: if the distribution curve was symmetrical these two figures would of course be the same as the average, but with such a skew distribution curve for latent heart block there is much divergence; in the other two groups the divergence is much less.

TABLE IV
AVERAGE AND MEDIAN P-R INTERVALS IN DIFFERENT GRADES OF HEART BLOCK

					P-R intervals (in seconds) in latent heart block		
					(IV) Latent block only	(III) With, at other times, dropped beats	(II) With, at other times, 2 : 1 or complete block
Average	0.239	0.278	0.254
Median	0.226	0.283	0.240
Mode	0.21	0.28	0.24
Quantiles	0.210-0.244	0.246-0.322	0.215-0.270
Usual range	0.21-0.24	0.27-0.32	0.19-0.26

The quantiles give the points between which half the cases lie; one-quarter being outside and above, and one-quarter outside and below. The usual range was chosen by inspection (before the quantiles were known) and has no exact mathematical meaning. It has been thought worth giving these additional figures because with such a wide range of variation average figures alone give a very imperfect picture.

CASES WITH THE LONGEST P-R INTERVALS

The ætiology of latent heart block in general has been discussed; in this section those with the longer P-R intervals will be considered in more detail.

Of the 31 cases where it was 0.26 sec. or above, 11 had rheumatic heart disease. They were on the whole young but not very young patients, at an age still liable to acute rheumatism rather than at the age most prone to suffer from the late results of old rheumatic heart disease. Only one was over 27 and the average age was just under 24 years. In 4 of the 11 the lengthening was transient and certainly due to an acute infection; in 3 it was transient and probably due to an acute infection (though in both these groups there was often a slightly

prolonged P-R interval before and/or after the transient increase); and in 4 it was persistent and not due to any active infection.

There were 12 of the 31 with myocardial disease, generally coronary atheroma or high blood pressure. They were, as might be expected, older patients, the average age being 58 and only three being under 50 years of age. The only two in whom the heart disease was not gross and obvious were *Case 34* (see p. 169) and *Case 44* who had developed high blood pressure after 10 years, but might have been described as normal as nothing was found at fault except his obesity and latent block (see p. 168).

One case had congenital heart disease (*Case 37*, see p. 169).

This left 7 cases where the heart was thought to be free from any organic disease. They were mostly young adults with an average age of 26 years. In 3 of the 7 no cause was found (*Cases 40, 49, and 51*; see p. 171). In the other 4 the long P-R interval were thought to be due to an attack of tonsillitis without any evidence that this was rheumatic. In 2 of these 4 the lengthened P-R disappeared quickly, confirming this view (*Cases 56 and 59*, see p. 170); but in 2, a recent follow-up has shown that it has persisted for 1 and 5 years respectively, and other features, the extreme sinus arrhythmia and the P-R interval of either about 0.36 or about 0.24 sec. suggest that possibly the attacks of tonsillitis were not the cause and that these were unusually marked effects of vagal activity (*Cases 42 and 47*, see p. 179).

This gives a very different picture, clinically and ætiologically, from that found with the higher grades of heart block. This is confirmed even by the most cursory glance at Table V. Here the 15 cases with a P-R interval of 0.30 sec. or more are shown: 9 were between

TABLE V
CLINICAL NOTES OF CASES WITH A P-R INTERVAL ABOVE 0.30 SEC.

Case No.	Sex and Age when first seen	Years under observation	P-R interval		Diagnosis of heart condition	Remarks
			longest	shortest		
45	f. 15	7	0.40	0.26	Rh. mitral stenosis	Lengthened by digitalis, but finally 0.36 without it.
49	m. 28	8	0.37	0.20	Normal	Extrasystoles and tuberculosis.
58	m. 77	3	0.37	—	B.P. 280/100	Stokes-Adams attacks; died after 3 years.
44	m. 40	9	0.36	0.31	Obesity	1919: obesity, B.P. 130/80; 1938: B.P. 180/120.
47	m. 20	1	0.36	0.24	Normal	Recent sore throat; ? incidental.
40	m. 43	10	0.35	0.16	Normal	Long P-R once only.
55	m. 52	1	0.35	0.35	Myocardial	Coronary atheroma; ? recent infarction.
57	m. 54	—	0.34	0.26	Rh. mitral stenosis	Congestive failure: increased by digitalis.
35	f. 24	1	0.34	0.14	Rh. aortic incompetence	? Rheumatic carditis but no other evidence.
42	f. 21	6	0.33	0.28	Normal	Recent sore throat when first detected.
50	f. 25	2	0.32	0.25	Rh. mitral st.	Recurrent carditis.
54	m. 24	—	0.32	0.19	Rh. mitral st.	During rheumatic fever.
48	f. 18	5	0.31	0.20	Rh. aortic incompetence	Mainly due to digitalis, but also recurrent rheumatism.
36	m. 14	6	0.31	0.21	Rh. mitral and aortic	Lengthened during terminal attack of rheumatic fever.
41	m. 68	2	0.30	0.25	Myocardial	Flutter with 4:1 block; digitalis restored N.R.

14 and 25 years of age; 7 had rheumatic valvular disease; 4 (or 5 if *Case 44* be included) were thought to have hearts that were free from organic disease, though 2 gave a history of sore throats; and only 3 were elderly men with signs of cardiac disease, such as are generally found with complete heart block.

There were also 12 cases, described in the paper as partial heart block with dropped beats (Campbell, 1943; most are listed in Table II on p. 56) who had at other times latent block only, with a P-R interval of 0.30 sec. or more. As a group they resemble the cases just described very closely: 8 were between 18 and 30 years of age; 5 had rheumatic heart disease; 5 were thought to be free from organic heart disease, but acute infections seemed a rather more important factor here; and only 2 were elderly patients (and in one of these the block was due to digitalis). In both these groups efforts have been made to keep in touch with most of the patients for many years, and only one has gone on to complete heart block (Case 58). Often the long latent block fell to a less prolonged figure (say, 0.21 to 0.25), but sometimes it persisted.

The patients with the highest grades of latent block are therefore a rather special group. In some, acute infections are the cause and the block is temporary; in others, the reason is obscure, and in some of these there seems little else the matter with the heart.

SUMMARY AND CONCLUSION

Latent heart block is a convenient term for a conduction time that is prolonged without any dropped beats or higher degree of block.

All those where the P-R interval was above 0.20 sec. have been included in this series. About 2 per cent of the patients sent to a cardiographic department showed this change, and 141 cases were analysed. The incidence fell very rapidly from 0.21 to between 0.24 and 0.25 sec., and then more slowly to 0.29, after which it was steadier, cases being seen at all levels up to 0.40 sec.; longer P-R intervals than this were very rare. In nearly half (46 per cent), the P-R interval was not more than 0.22 sec. In nearly one-quarter (22 per cent), it was 0.26 sec. or more.

In addition, 29 cases, which sometimes had dropped beats and sometimes latent block only, and another 27, which sometimes had 2 : 1 and/or complete heart block and sometimes latent block only, were analysed.

When there was latent block only, the P-R interval was most commonly between 0.21 and 0.24 sec. When latent block interrupted complete and/or 2 : 1 block the figure was much the same with a rather wider common range, 0.19 to 0.26 sec. On the other hand, when there were at times dropped beats, and at other times latent block only, the P-R interval was on the average longer, and was generally from 0.26-0.32 sec., possibly because more of these cases were due to a transient acute infection.

As a rule, latent block did not progress to higher grades of heart block. In many it diminished as the effect of an acute infection disappeared; in some it remained at a fairly constant level; and in some it was found, occasionally or from time to time interrupting complete and/or 2 : 1 heart block.

The ætiology varied somewhat in the different groups. Where the P-R interval was from 0.20 to 0.25 sec., all types of heart disease were represented in much the same proportions as might be found in any collection of cardiac cases.

Where the P-R interval was 0.26 or above, the ætiology was more like that found in cases with dropped beats: 41 per cent (as against 11 per cent with a P-R interval from 0.20 to 0.25 sec.) had acute rheumatism or other active infections, mostly tonsillitis, and there were fewer cases with thyrotoxic or normal hearts.

Cases with latent block and at other times 2 : 1 or complete block were ætiologically like the cases with complete heart block, i.e. older patients with atherosclerosis or primary myocardial disease (69 per cent against 26 per cent in the two previous groups, or 86 per cent against 39 per cent if hyperpietic cases were included).

Acute rheumatic carditis was a common cause of P-R intervals that were much prolonged, to 0.26 sec. or above. Chronic rheumatic heart disease occasionally caused these longer

values, but was more often responsible for the slighter increases from 0.20 to 0.24 sec. Other acute infections sometimes produced quite a long P-R for a time; these including attacks of tonsillitis that were almost certainly not rheumatic.

Latent heart block, especially of the lesser grades, was seen in all types of chronic myocardial disease. Even here it seemed rare for it to progress gradually to complete heart block, though often it interrupted complete or 2 : 1 block, sometimes after surprisingly long intervals.

Both in thyrotoxicosis and myxœdema, long P-R intervals were observed, and the relationship of this to iodine therapy in thyrotoxicosis needs more investigation.

No instances of latent block due to trauma were included in the series, though such cases have been reported; and one due to blast has been mentioned.

There were no cases where latent block could with certainty be attributed to diphtheria, but such a history was noted in some cases as a possible cause that could not be excluded.

Some curious cases have been described in which P-R intervals, even up to 0.30 sec. or more, were found without any other evidence of heart disease. As some of these persisted they did not seem to be due to infection, and it is suggested that exceptionally overaction of the vagus may produce unusually long P-R intervals. In some, which have been described rather fully, the longest P-R intervals were irregular or intermittent.

Some few cases with a P-R interval up to 0.22 sec. or even higher seemed to be normal in every way.

APPENDIX OF CASE NOTES

Cases with Acute Rheumatism

Some typical cases have been referred to in the text (p. 167) and in a previous paper (Campbell, 1943, p. 56). A less usual case is added below.

Case 35. C., aged 24, complained that she had fainted after a hot bath; probably her doctor would not have sent her to hospital had he not found a murmur. She had free aortic regurgitation with some enlargement of the heart, and though there was no rheumatic history slight mitral stenosis confirmed that it was rheumatic. Her attack seemed like an ordinary faint and had none of the characteristics of a Stokes-Adams attack so that a P-R interval of 0.34 sec. was a surprise. We expected this would prove to be a permanent feature of her heart, but she was told to lead an easy life and return in two weeks in case a change should show there had been active carditis.

She resumed her normal life and was not seen again till she was written for after six months. She had no symptoms except the slight degree of dyspnœa to which she was accustomed. Her P-R interval was now 0.14 sec. This gives an important indication of the changes that may be going on in the heart unsuspected, and means that even in the young an attack of faintness may occasionally indicate cardiac damage.

Cases with Chronic Rheumatic Heart Disease

Several of these have been quoted shortly in the text, and the following is given in more detail because of the unusual length of the P-R interval.

Case 45. I. J. had chorea and rheumatism at 11, and four years later (1932) was found to have enlargement of the heart (13 cm./22 cm.) and high-grade mitral stenosis. She complained of cough and dyspnœa and was unusually cyanosed. The P-R interval was prolonged, and on 12 occasions during the next year it varied between 0.24 and 0.28 sec. Her heart rate was generally 108-120, but there was no other evidence of recurrent rheumatism.

During the next two years, treatment with digitalis improved her condition as regards her dyspnœa and cyanosis, but the heart rate remained about 96-108. It lengthened the P-R interval to between 0.32 and 0.40 sec. Although she was seen nearly every fortnight during three years no dropped beat was ever observed (Campbell, 1942, p. 140).

In 1934 she started work and was seen less regularly. In 1936 some œdema of the feet developed and she began having short paroxysms of tachycardia, lasting up to half an hour. She was never quite so well after this, but continued at easy work with short periods in hospital for another two years. The P-R interval varied between 0.32-0.36 sec. and was often this length when she was not taking digitalis. She was lost sight of after December 1937, and died elsewhere in February 1939 and we have not been able to get details of her final illness.

Here a P-R interval of 0.28-0.36 sec. persisted for at least 7 years, and even when she was taking digitalis no dropped beats were ever observed.

*Cases with Myocardial Disease**Case 41. Latent block, producing spontaneous 4 : 1 block when there was auricular flutter.*

A man, aged 68, with a large heart and a blood pressure of 160/100 came to hospital, complaining of cough and dyspnoea. His pulse rate was 76, but this was found to be due to auricular flutter with spontaneous 4 : 1 block, which after exercise changed to irregular 2 : 1 block. He was given Nativelle's digitaline, and when he came to hospital a fortnight later said he had taken 1/600 grain t.i.d. the first week, and q.i.d. the second week. This had restored normal rhythm and the P-R interval was 0.30 sec., compared with his normal of 0.24, which was found during the months following when he was not taking digitalis. In this case the latent heart block was as helpful as digitalis treatment would have been in producing such a degree of block that the ventricular rate was normal as long as he was not exerting himself.

This is not uncommon with auricular fibrillation where some natural degree of block in an elderly patient helps in the same way that digitalis would by reducing the ventricular rate; but with fibrillation it cannot be demonstrated so elegantly as in this case of flutter.

Case 44. Latent block and obesity, followed after some years by high blood pressure.

A man, aged 40, was admitted to hospital for fatigue, dyspnoea, and headaches, the latter due to severe myopia in the right eye. All his symptoms were attributed to his excessive weight of 16 stone, and nothing else abnormal was found except a P-R interval of 0.34-0.36 sec. (Fig. 2). The Wassermann reaction was negative. He improved with thyroid and dieting.

Two years later his condition was somewhat better as he had kept his weight lower, but the P-R interval was still 0.36 sec. He had taken no thyroid for some time as it upset him, but had restricted his diet.

After another eight years he was re-admitted for increasing dyspnoea. He had never felt well but he had been at work all the time and had kept his weight to 15 stone by eating very little except bread and butter. The P-R interval was 0.31 sec. and the blood pressure, which had been 130/80 in 1929 and 145/95 in 1931, had risen to 180/120; the first sound was reduplicated. No other abnormality was found, except that the cardiogram now showed flat T waves as well as the latent heart block.

Case 55. Latent block and angina, with anginal pain felt in a phantom arm.

One of these patients is of interest, not only because of his long P-R, 0.35, but because he had anginal pain in a phantom arm. His right arm was blown off in France in 1917. Eighteen years later, in May 1935, he began having anginal pain which started across the chest and spread down the right phantom arm with great regularity. In October 1935 his pain became worse and he may have had a cardiac infarct, but there was no cardiographic proof of this. In May 1936, when he was 52, he came to hospital as his angina was troublesome and persistent. Nothing was found on examination except a P-R interval of 0.35 sec. which remained constant for the three months he was under observation. He had had diphtheria in 1933.

We were much interested in the pain he felt in his phantom arm because of its occurrence so long after the loss of his arm, but not surprised as it seemed to us that with a pain that was normally referred from the centre to the arm, it must still be felt there if the arm was able to give rise to sensations of any sort: we did not realize till seeing the paper by Cohen and Wallace Jones (1943) that such a case had not been reported. Dr. S. Suzman made many attempts to study his reaction to nitrites, but it was not easy to provoke the pain by any exercise he took while under observation as the onset was less regular than in most cases of angina.

Case 58. Stokes-Adams attacks: latent block only while under observation.

A fine, healthy-looking old seaman, aged 76, had a Stokes-Adams attack. He had no other complaints except a little dyspnoea. A year later, although he had no more attacks, he was sent to hospital and the cardiogram showed a P-R interval of 0.37 sec. He gave a clear account of his pulse getting slower with more dropped beats, and stated that the pulse rate often fell to 33, especially in bed in the morning, so that it is almost certain he had complete or 2 : 1 heart block. His heart was enlarged, his blood pressure 280/100, and the Wassermann reaction positive. He was not seen again and died suddenly three years later.

Cases with Tonsillitis

Case 5 and *Case 17* have been reported already (Campbell, 1943, p. 57) as at one stage they had partial heart block with dropped beats, but are pertinent here as at a later stage they had latent heart block only.

Case 59. S. S., aged 18, had tonsillitis, a first attack, and was at home for a week without seeing his doctor. He returned to work but did not feel as well as usual. After two weeks at work he woke up feeling sick, and on his way to the lavatory fell down unconscious and cut his face severely. His doctor found his pulse rate 34, and kept him in bed till he came to hospital four days later.

Nothing was found abnormal except an altered first sound, slight albuminuria, and a P-R interval of 0.28 sec. (Fig. 3A); a week later this was 0.18, and after another week 0.16 sec. (Fig. 3B). He felt quite well and continued at work. It seems unlikely that this was an undiagnosed attack of diphtheria.

If he had not been seen again this would seem a simple case where the P-R interval was lengthened by tonsillitis and where there had been complete heart block with a Stokes-Adams attack, his recovery being complete and rapid. But nine months later when asked to come to hospital again he had a curious cardiogram. He had been quite well until "influenza" a month before and had had no more attacks of faintness or loss of consciousness. The usual P-R interval was now 0.23 sec., but sometimes it was much shorter as though there was ventricular escape (R_4 in Fig. 7D and to a lesser extent

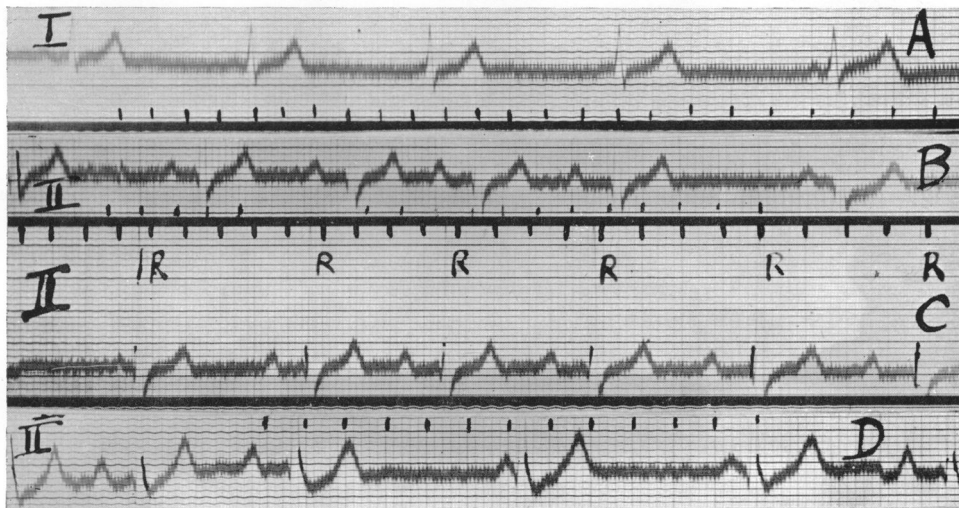


FIG. 7.—Sinus arrhythmia with variation of the P-R interval, which is at times much below its usual length of 0.23 sec., probably due to ventricular escape. From the same Case 59 as Fig. 3, all taken nine months later (17/2/33), shortly after an attack of influenza.

- (A) Ventricular escape (see text).
- (B) Shorter P-R interval after a pause in the sinus rhythm.
- (C) Ventricular escape (R_1), nearer to the auricular wave than in (A).
- (D) Sinus arrhythmia with ventricular escape twice during the slower period.

R_4 of Fig. 7B). Sometimes this happened where there was a longer pause, probably during the expiratory phase of sinus arrhythmia. Sometimes, however, it happened without any sinus arrhythmia (see Fig. 7A): there the P-P intervals were constant, but the R-R intervals were shorter so that the first four P-R intervals became shorter or non-existent, until the fifth R-R interval being longer allowed P-R to reappear; nodal rhythm arising in different sites could explain this, but the regular P-P rate makes this unlikely, and frequent ventricular escape at a rather rapid rate seems the most likely explanation.

Cases with Tonsillitis that may have been merely incidental, in which event they should be included as Cases with no other evidence of organic heart disease. Cases with no other evidence of heart disease have been described in the text (pp. 170-172).

Case 42. A nurse, aged 21, was admitted with a sore throat, a temperature of 102, and a pulse rate of 80. The third day she felt well and her temperature was normal, but her heart rate was found to be irregular and about 50. Dropped beats were suspected and two days later the P-R interval was 0.29-0.32 sec. (Fig. 8). A week later it was unchanged, and three weeks after when she had been feeling perfectly well, a record was obtained that was at first thought to be dropped beats but was sino-auricular block or perhaps extreme sinus arrhythmia as the long P-P interval was much less than twice the normal: no progressive lengthening of the P-R interval of 0.32 sec. was observed. A week later when she was allowed up, P-R was again 0.30 sec. and regular.

She was not seen again for two months as she was on holiday, feeling quite well. Four months after the sore throat that had lasted only two days, sino-auricular block or extreme sinus arrhythmia with other periods regular was again observed, with the P-R interval unchanged; in the absence of symptoms or other signs she was allowed to resume work.

After another year, during which she had continued at work, she felt well and showed no physical signs, except that P-R was still 0.30 sec. She gave no history of rheumatism at any time. It was at that time thought unlikely that she had any degree of heart block before her short illness.

During the next four years she carried out her work as a nurse without any symptoms or disability,

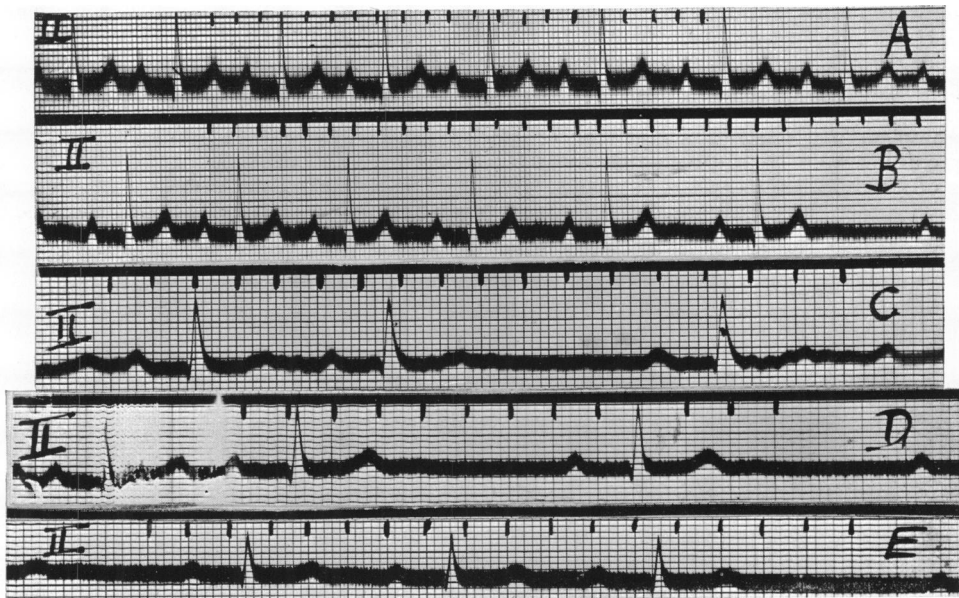


FIG. 8.—Extreme sinus arrhythmia with a long P-R interval, persisting for at least five years. Case 42.

- (A) The most usual finding with a rate that is nearly regular (shortest R-R, 0.86; longest R-R, 0.96).
 (B) On the same date, changing to a much slower rate—the six P-P's being 0.92, 0.92, 1.02, 1.07, 1.18, and 1.54 sec. (C), (D), and (E) all show marked sinus arrhythmia, the last two being taken during deeper respiration.

(C) Was taken on 14/2/38, three weeks after a sore throat (which may have been incidental); all the others were taken on 2/3/39.

In 1943 the P-R interval was still 0.31; the rate was regular when recorded, though at other times there was some irregularity that did not appear to be associated with respiration.

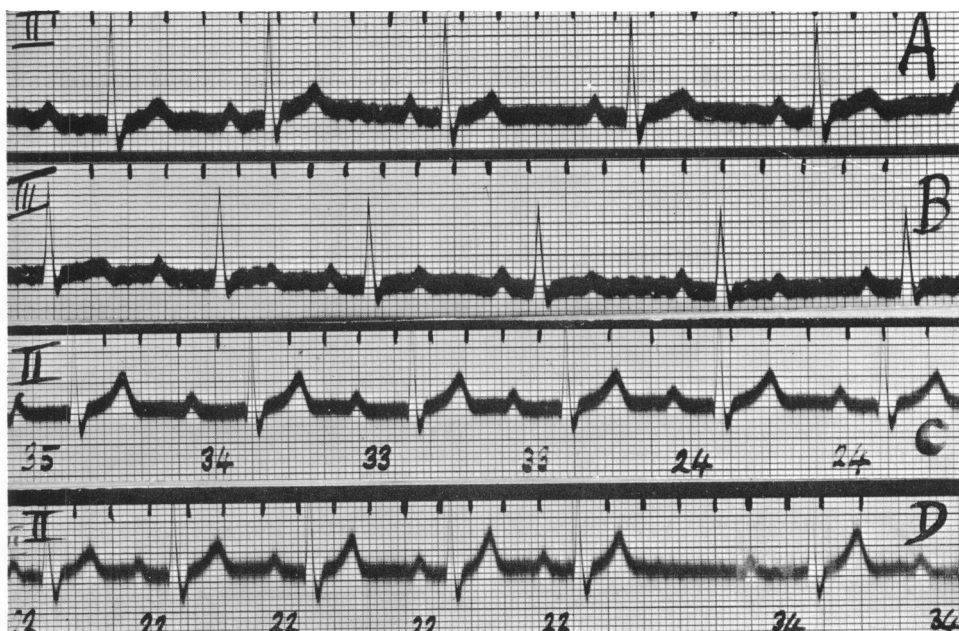


FIG. 9.—Changes of long and shorter P-R intervals. Case 47; see also Fig. 1.

- (A) and (B) Both show the change from the long to the shorter P-R interval at the beginning of the picture (5/6/39.)
 (C) Shows better the change from the long to the shorter P-R interval; the P-P intervals are nearly constant, but shorten a little from 0.96 to 0.87 sec. (26/6/39.)
 (D) Shows the change from the shorter to the long P-R interval; the last P-P interval before the long P-R is much longer than usual (but not twice as long); as a rule, however, the rate of the heart with the long or with the shorter P-R interval was the same. (26/6/39.)

leading a normal life in every way. Nevertheless, her P-R interval was still 0.31 sec. as it had been four years before. Her heart rate was between 56 and 64 with some slight irregularity of the sinus type that was not, however, associated with respiration. There were no abnormal physical signs.

At first she was thought to be a case of partial heart block following tonsillitis as in the two cases described with dropped beats (Cases 5 and 17), one of these also being a nurse; but its long persistence without any other symptoms and the association with sinus arrhythmia that was at times extreme enough to simulate sino-auricular block suggests that it may be a physiological, though unusual finding in a patient with very high vagal tone.

Case 47. A young man, aged 20, came to hospital complaining of breathlessness and pain in the left side of the chest which had been present most days for a year. He was anxious about tuberculosis as he had a bad family history, but clinically and on X-ray examination his chest was normal. He was liable to sore throats and had been ill with this three weeks before for four days, but had not seen his doctor as it seemed no worse than usual. He had fainted once nine months before. He gave no history of rheumatism, but had had diphtheria as a child.

Nothing was found on examination except that his P-R interval was about 0.24 sec. For this reason only he was asked to return the following week, when his P-R interval was sometimes 0.24 and sometimes 0.34 sec. This was observed on many occasions during the next nine months, the longer P-R interval always being between 0.32 and 0.36, and the shorter always between 0.22 and 0.26 sec. On one occasion only he was seen with a heart rate nearly 100 and the interval was as short as 0.20 sec.

Generally the rate was relatively slow, about 60, and sometimes as slow as 44. There was no general difference in the heart rate whether the P-R interval was about 0.24 or about 0.34 sec., the average rate in both cases being 61. On one occasion (see Fig. 9D) there was a change to the long P-R interval after a pause and perhaps with a slower rate afterwards, but on the same day another plate showed the reverse change without any change of rate (see Fig. 9C). Most often, all the beats in one lead showed the same P-R interval, but records showing the change over have been picked out for illustration.

During the year he was under observation there was no regular change in his symptoms, which were sometimes better and sometimes worse. His sedimentation rate was normal; an X-ray of his chest was clear; and the throat surgeon reported that though he had frequent slight attacks of tonsillitis there was no focal sepsis. He was not unduly thin and was putting on weight.

At first the long P-R interval was attributed to his recent tonsillitis, but the relatively slow heart with the variable P-R interval suggest a high degree of vagal tone, and it is possible that this was usual with him and quite independent of the recent attack. Attempts to trace him recently have not been successful.

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